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☑	USPT	c/ebp	YES	ADJ	ASSIGNEE	L4
☑	USPT	hepatic lipase promoter$1	YES	ADJ	ASSIGNEE	L5
☑	USPT	hl promoter	YES	ADJ	ASSIGNEE	L6
☑	USPT	HL promoter$1	YES	ADJ	ASSIGNEE	L7
☑	USPT	hepatic lipase	YES	ADJ	ASSIGNEE	L8
☑	USPT	L8 and (L4 or L3) and L2 and L1	YES	ADJ	ASSIGNEE	L9
☑	USPT	L8 and (L4 or L3) or L2	YES	ADJ	ASSIGNEE	L10
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09/926,944

SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2003/May W4
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File 155:MEDLINE(R) 1966-2003/May W4
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***File 155: Medline has been reloaded and accession numbers have** changed. Please see HELP NEWS 155.

Set	Items	Description
S1	42034	ESTROGEN(W)RECEPTOR?
S2	2339	HEPATIC(W)LIPASE?
S3	6671	P300 OR CEBP?
S4	0	S1 AND S2 AND S3\
S5	0	S1 AND S2 AND S3
S6	125982	TRANSCRIPTION(W)FACTOR?
S7	0	S1 AND S2 AND S3
S8	1	S1 AND S2 AND S6

8/9/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09966961 21891004 PMID: 11893774
Estrogen receptor **-mediated repression of human** hepatic lipase **gene transcription.**
Jones Daniel R; Schmidt Robert J; Pickard Richard T; Foxworthy Patricia S ; Eacho Patrick I
Lilly Research Laboratories, Cardiovascular Research Division, Eli Lilly and Company, Indianapolis, IN 46285, USA.
Journal of lipid research (United States) Mar 2002, 43 (3) p383-91, ISSN 0022-2275 Journal Code: 0376606
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Estrogen replacement therapy in women decreases **hepatic lipase** (HL) activity, which may account for the associated increase in HDL cholesterol. To investigate whether estrogen decreases HL transcription, transient cotransfection assays with HL promoter and **estrogen receptor** -alpha (ERalpha) expression constructs were performed in HepG2 cells. 17beta-estradiol (E(2)) decreased transcription driven by the -1557/+41 human HL promoter by up to 50% at 10(-7) M. Mutation of ERalpha by deletion of its transactivation domains or ligand-binding domain eliminated E(2)-induced repression of the promoter, whereas deletion of the DNA-binding domain of ERalpha resulted in a 7-fold activation by E(2). The E(2)-induced repression was maintained after mutation of a potential estrogen-response element in the promoter. The region of estrogen responsiveness was localized to -1557/-1175 of the HL promoter by deletion analysis. Mutation of an AP-1 site at -1493 resulted in a partial loss of E(2)-induced repression, similar to that caused by deletion of nucleotides -1557 to -1366. Gel shift assays with nuclear extracts from E(2)-treated HepG2 cells stably expressing ERalpha demonstrated an increase in binding to an AP-1 consensus oligonucleotide. The AP-1 activator, phorbol 12-myristate 13-acetate, inhibited the HL promoter by greater than 50%. Collectively, the data suggest that estrogen represses the transcription of the HL gene, possibly through an AP-1 pathway.

NCBI PubMed National Library of Medicine NLM

PubMed Nucleotide Protein Genome Structure PMC Taxonomy OMIM B

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#3	Related Articles for PubMed (Select 11893774)	16:59:06	250
#1	Search journal of lipid research[jour] AND 43[volume] AND 383[page] Field: Title Word	16:58:26	1

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09/924,944

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1: Atherosclerosis 2001 Feb 15;154(3):625-32 Related Articles, Li

ELSEVIER SCIENCE FULL-TEXT ARTICLE

Hepatic lipase promoter activity is reduced by the C-480T and G-216A substitutions present in the common LIPC gene variant, and is increased by Upstream Stimulatory Factor.

Botma GJ, Verhoeven AJ, Jansen H.

Department of Biochemistry, Cardiovascular Research Institute (COEUR), Erasmus University Rotterdam, PO Box 1738, 3000 D Rotterdam, The Netherlands.

The common -216G-->A and -480C-->T substitutions in the promoter region of the human hepatic lipase (LIPC) gene show high allelic association, and are correlated with decreased hepat lipase activity and increased high-density lipoprotein cholesterc levels. To test the functionality of these substitutions, CAT-reporter assays were performed in HepG2 cells. LIPC (-650/+48) but not (-650/+61) promoter constructs showed transcriptional activity. LIPC (-650/+48) constructs with both -216A and -480T exhibited significantly lower promoter activity (-45%) than the wild-type form. Activities of -289/+48 constructs were not significantly affected by the -216G-->A substitution. The -480C/T site lies within a binding region for Upstream Stimulatory Factor (USF). Gel-shift assays showed th the binding affinity of USF protein for HL specific oligonucleotides was decreased four-fold by the -480C-->T substitution. However, promoter activity of the -650/+48 constructs was not significantly affected by the -480C-->T substitution alone. Co-transfection of HepG2 cells with USF(43) cDNA yielded a similar dose-dependent increase in activity of a -650/+48 constructs; the absolute difference in promoter activi increased but the relative difference between the variant promoter forms was maintained. Our studies demonstrate that th common LIPC promoter variation is functional, which explains th

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1: Biochem Soc Trans 1997 Nov;25(4):S689 Related Articles, Li

Regulation of hepatic lipase expression in HepG2 cells.

Nimmo L, McColl AJ, Rosankiewicz JZ, Richmond W, Elkeles RS.

Unit of Metabolic Medicine, Imperial College School of Medicine at St Mary's, London, UK.

PMID: 9450116 [PubMed - indexed for MEDLINE]

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1: Eur J Biochem 1997 Jul 1;247(1):148-59

Structural and functional analysis of the promoter of the hepatic lipase gene.

Chang SF, Scharf JG, Will H.

Heinrich-Pette-Institut fur experimentelle Virologie und Immunologie an der Universitat Hamburg, Germany.

Hepatic lipase (HL) gene transcription is almost exclusively limited to hepatocytes. Here we have studied sequences and transcription factors regulating basal and hepatocyte-restricted HL promoter activity. Sequencing of a cloned 3.4-kb HL promoter fragment revealed three Alu repeat sequences and a consensus hepatocyte-enriched nuclear transcription factor 1 (HNF1) binding site located upstream of one major and one minc transcription initiation site. By transfection of cell lines of hepat and non-hepatic origin and of primary hepatocyte cultures, sequences controlling basic HL promoter activity and negative elements located downstream and upstream thereof which extinguish or enhance this activity were defined. Some HL-promoter fragments with internal deletions were active only in primary hepatocyte cultures. Human HNF1 protein was shown t bind to the HL-specific HNF1 response element and the activity of a heterologous promoter was enhanced by HL-HNF1 in rat primary hepatocyte cultures but not in the context of the authentic 3.4-kb HL promoter sequences. In cell lines the presence of HNF4 but not of HNF1 and vHNF1 mRNA was found to correlate with HL gene expression although no perfect consensus HNF4 binding motif was detected in the promoter region tested. Taken together, these data indicate that hepatocyte-specific HL gene transcription is controlled by positive and negative transcription regulatory proteins which bir to sequence motifs within and outside of the proximal 3.4-kb promoter fragment studied. For the elucidation of the control of HL promoter activity in vivo the use of primary hepatocyte

cultures is essential.

PMID: 9249021 [PubMed - indexed for MEDLINE]

1: Gene 1996 Nov 21;180(1-2):69-80

Transcription of the human hepatic lipase gene is modulated by multiple negative elements in HepG2 cells.

Oka K, Ishimura-Oka K, Chu MJ, Chan L.

Department of Cell Biology, Baylor College of Medicine, Housto TX 77030, USA.

The expression of the hepatic lipase (HL) gene is highly tissue specific. In order to identify cis-acting elements which regulate the expression of this gene in the liver, multiple deletion mutant of the 5'-flanking region of the HL gene fused to the human growth hormone gene were transfected in HepG2 cells, which normally produce HL. Transient expression assays indicated the presence of negative (at nucleotides (nt) -1576(/)-1342 and -6 (/)-407) and positive (at nt -1862(/)-1576 and -50(/)-9) regulatory elements. Transfection of HeLa cells, which do not produce HL, with the same deletion constructs resulted in a similar pattern of promoter activities. However, additional negative (nt -138/-50) and positive (nt -407(/)-138) elements were found. DNase I footprint analysis of the proximal and dista HLpromoter sequences with HepG2 and HeLa cell nuclear extracts identified seven protected regions: A, nt -1540(/)-152 B, -1505(/)-1473; C, -1467(/)-1460; D, -592(/)-577; E, -565(/ 545; F, -234(/)-220; and G, -70(/) -48. Sites A, B, C, D and E were located within regions containing negative regulatory elements. In order to determine which nuclear factor interacts with the negative elements, sites B, D and E were mutated and the effects of mutation on competition in a gel retardation assay and on promoter activity were studied. When the binding motif f AP1 in sites B, D and E was mutated, the specific DNA-protein complexes were not competed with the mutant oligonucleotides and promoter activity increased twofold. The magnitude of the increase is less than expected from the deletion analysis, and simultaneous mutations did not cause further increase in

promoter activity, which suggests that other sites are involved i this negative modulation. These results suggest that the transcription of the HLgene in HepG2 cells is negatively modulated by multiple cis-acting negative elements and AP1-lik nuclear factor may play some role in this modulation.

PMID: 8973349 [PubMed - indexed for MEDLINE]
